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EXAMINER

HABTE, KAHSAY

ART UNIT	PAPER NUMBER
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1624

DATE MAILED: 10/25/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

**Office Action Summary**

Application No.

10/715,358

Applicant(s)

HOLDER ET AL.

Examiner

Kahsay Habte, Ph. D.

Art Unit

1624

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

- 1) ☐ Responsive to communication(s) filed on \_\_\_\_.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

- 4) ☒ Claim(s) 1-29 is/are pending in the application.
- 4a) Of the above claim(s) \_\_\_\_ is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 1-7 and 9-29 is/are rejected.
- 7) ☒ Claim(s) 8 is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_ are subject to restriction and/or election requirement.

**Application Papers**

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

**Priority under 35 U.S.C. § 119**

- 12) ☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☒ All b) ☐ Some \* c) ☐ None of:
1. ☒ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

**Attachment(s)**

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)  | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date. ____. |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)                                   | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152)             |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)<br>Paper No(s)/Mail Date ____. | 6) <input type="checkbox"/> Other: ____.  |

### DETAILED ACTION

1. Claims 1-29 are pending in this application.

#### ***Double Patenting***

2. A rejection based on double patenting of the "same invention" type finds its support in the language of 35 U.S.C. 101 which states that "whoever invents or discovers any new and useful process ... may obtain a patent therefor ..." (Emphasis added). Thus, the term "same invention," in this context, means an invention drawn to identical subject matter. See *Miller v. Eagle Mfg. Co.*, 151 U.S. 186 (1894); *In re Ockert*, 245 F.2d 467, 114 USPQ 330 (CCPA 1957); and *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970).

A statutory type (35 U.S.C. 101) double patenting rejection can be overcome by canceling or amending the conflicting claims so they are no longer coextensive in scope. The filing of a terminal disclaimer cannot overcome a double patenting rejection based upon 35 U.S.C. 101.

3. Claims 1-8 and 27-29 are provisionally rejected under 35 U.S.C. 101 as claiming the same invention as that of claims 1-8 and 26-28 of copending Application No. 10/715,556. This is a provisional double patenting rejection since the conflicting claims have not in fact been patented.

***Claim Rejections - 35 USC § 102***

4. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

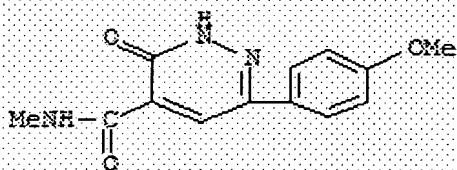
A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1-7 and 27-29 are rejected under 35 U.S.C. 102(b) as being anticipated by Yoshizaki et al. (WO 99/44995). Cited reference discloses a compound of interest: 2,3-dihydro-6-(4-methoxyphenyl)-N-methyl-3-oxo-4-Pyridazinecarboxamide that is the same as applicants when applicant's formula (I) has the following substituents:

Ar = phenyl substituted with methoxy; A = CONH-methyl (see below).

RN 243862-95-5 CAPLUS  
CN 4-Pyridazinecarboxamide, 2,3-dihydro-6-(4-methoxyphenyl)-N-methyl-3-oxo-  
(SCI) {CA INDEX NAME}



***Claim Rejections - 35 USC § 112***

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 17-26 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for the treatment of cranial and spinal traumas and

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peripheral neuropathies, obesity, type II diabetes, atherosclerotic cardiovascular diseases, essential hypertension, polycystic ovary syndrome, does not reasonably provide enablement for the rest of the diseases. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims. It is recited for example in claim 17 a method of treating neurodegenerative diseases, strokes, metabolic diseases, syndrome X, immunodeficiency and cancer, but the specification is not enabled for such a scope.

A number of factors are relevant to whether undue experimentation would be required to practice the claimed invention, including "(1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims." In re Wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988).

(1). Breadth of Claims:

(A) - The scope of use that applicants intend to claim is very broad.

**Metabolic diseases**

Disorders may affect metabolism, which is how the body processes substances needed to carry out its functions. Such disorders are often caused by genetic abnormalities that

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result in the absence of a specific enzyme needed to stimulate a metabolic process.

Depending on the disorder, the effects may be serious or fairly harmless.

There are several types of metabolic disorders: Carbohydrate Metabolism Disorders, Pyruvate Metabolism Disorders, Aminoacid Metabolism Disorders, etc.

### Carbohydrate Metabolism Disorders

Carbohydrates are sugars. Many sugars besides the well-known glucose, sucrose, and fructose are present in foods. Some sugars, such as sucrose, must be processed (metabolized) by enzymes in the body before they can be used as a source of energy. If the enzymes needed to process them are missing, these sugars can accumulate, causing problems. Two examples are:

**Galactosemia** (a high blood level of galactose) is usually caused by the lack of galactose 1-phosphate uridyl transferase, one of the enzymes necessary for metabolizing galactose. This disorder is present from birth.

**Hereditary fructose intolerance** is a hereditary disorder in which the body cannot use fructose because the enzyme phosphofructaldolase is absent. As a result, fructose 1-phosphate, a by-product of fructose, accumulates in the body, blocking the formation of glycogen and its conversion to glucose for use as energy.

### Pyruvate Metabolism Disorders

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Pyruvate is formed in the processing of carbohydrates, fats, and proteins. Hereditary problems with the processing of pyruvate can cause a wide variety of disturbances.

Pyruvate is an energy source for mitochondria, the energy-generating components of a cell. A problem with pyruvate metabolism can disturb the functioning of the mitochondria, causing any of a variety of symptoms, such as muscle damage, mental retardation, seizures, a buildup of lactic acid leading to excess acid in the body (acidosis), or failure of organ function, including that of the heart, lungs, kidneys, or liver. Such problems may develop any time between early infancy and late adulthood. Exercise, infections, or alcohol consumption can worsen symptoms, leading to severe lactic acidosis with muscle cramping and weakness. Two examples are:

***A deficiency of the pyruvate dehydrogenase complex***, a group of enzymes needed to process pyruvate, results in insufficient levels of acetyl coenzyme A, which is essential for energy production. The major symptoms include slowed muscle action, poor coordination, and a severe balance problem that makes walking nearly impossible. In addition, seizures, mental retardation, and brain malformation may occur. This disorder cannot be cured, but some people are helped by a diet high in fat.

***Absence of pyruvate carboxylase***, an enzyme, interferes with or blocks the production of glucose in the body. Lactic acid and ketones build up in the blood, causing nausea and vomiting. Often this disease is fatal. The synthesis of amino acids, the building blocks of proteins, also depends on pyruvate carboxylase. When this enzyme is missing, the production of neurotransmitters (substances that transmit nerve impulses)

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is reduced, leading to a variety of neurologic symptoms, including severe mental retardation. Low blood sugar levels (hypoglycemia) and the buildup of acids in the blood (acidosis) may be relieved by eating frequent carbohydrate-rich meals, but no replacements for the missing neurotransmitters are available to treat the neurologic symptoms.

### Amino Acid Metabolism Disorders

Amino acids, the building blocks of proteins, have many functions in the body.

Hereditary disorders of amino acid processing can be defects in either the breakdown of amino acids or their transport into cells. Many of these disorders, including phenylketonuria, have been identified.

**Phenylketonuria** (*PKU*, *phenylalaninemia*, *phenylpyruvic oligophrenia*) is a hereditary disorder in which the enzyme that processes the amino acid phenylalanine is missing, resulting in a dangerously high level of phenylalanine in the blood.

Phenylalanine is normally converted to tyrosine, another amino acid, and eliminated from the body. Without the enzyme that converts it, phenylalanine builds up in the blood and is toxic to the brain, causing mental retardation.

There are other metabolic disorders, which affect e.g. calcium and phosphorus as well as other elements in the body.



## **Cancer**

The scope of use that applicants intend to claim is very broad. To this day, it is impossible to treat all cancer cells with a single pharmaceutical drug. Please see below for the explanations that cancer cells are broad and different one from the other.

Cancer cells can exist in different parts of the body and the nature of these cancer cells differs one from the other. For example, the treatment of bone cancer cannot be the same as the treatment of skin cancer. The drug that inhibits bone cancer cells may require more doses than the cancer cells in skin. The form of delivery for both said cancers (radiation, ointment, tablets, etc.) is not the same. For instance, one has to get deep to the bones to inhibit the cancer cells in the bones, while applying the drug on the surface of the skin can inhibit cancer cells on skin. It is also a fact that some cancer cells need more drugs than the others. It is also true that the compounds could be having antagonistic effect or agonistic effect when administered to the body. Which diseases (cancer cells) are inhibited by the administration of the drug and which are not? Applicants claim that all cancer cells can be treated by single pharmaceutical drugs, thus is not enabled.

It can be shown that cancer cells in general are extraordinarily broad. For a compound or genus to be effective against cancer cells generally is contrary to medical science. Cancer is a disease, which can take place in virtually any part of the body. There is a vast range of forms that it can take, causes for the problem, and biochemical pathways

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that mediate cancer. There is no common mechanism by which all, or even most, cancers arise. Accordingly, treatments for a cancer or inhibition of cancer cells are normally tailored to the particular type of cancer cells present, as there is no, and there can be no “magic bullet” against cancer cells generally.

Even the most broadly effective antitumor agents are only effective against a small fraction of the vast number of different cancers known. This is true in part because cancers arise from a wide variety of sources, such as viruses (e.g. EBV, HHV-8, and HTLV-1), exposure to chemicals such as tobacco tars, genetic disorders, ionizing radiation, and a wide variety of failures of the body's cell growth regulatory mechanisms. Different types of cancers affect different organs and have different methods of growth and harm to the body, and different vulnerabilities.

### **Neurodegenerative diseases**

It has been recited in claims 17-25, a method of treating neurodegenerative diseases. Neurodegenerative disorders are extremely varied in origin and nature of effect. The origin and the nature of many neurodegenerative disorders such as Huntington's disease, Pick's disease, Frontotemporal dementia, Cerebro-Oculo-Facio-Skeletal (COFS) syndrome (cranofacial and skeletal abnormalities), Motor neuron disease (muscle weakness), Corticobasal ganglionic degeneration, Creutzfeldt-Jacob disease (fatal disease), Dementia with Lewy bodies, and Progressive supranuclear palsy

Dementia are different one from the other. Many neurodegenerative disorders are untreatable to this day.

The symptoms and nature of these diseases are also different one from the other. It can be shown that many of these neurodegenerative disorders have different origin and nature of effect. Some neurodegenerative disorders are hereditary (Charcot-Marie-Tooth disease). Many neurodegenerative disorders vary in how they affect the body and its functions. Diseases such as Cerebral palsy, and Parkinson's disease affect the movement of the patient. Diseases such as Alzheimer's disease affect the memory of the patient.

### **Stroke**

Stroke represents one of the most intractable medical challenges. Stroke is estimated to cause about 15% of deaths, behind only heart disease and cancer. Even those who survive normally suffer from persistent damage, including motor and speech disturbances and/or convulsions. Despite a tremendous effort to resolve these problems, cerebrovascular therapy as so far been limited to trying to prevent further damage in areas on the margins of the ischemic focus, thus trying to maintain adequate perfusion in remaining intact areas, and thereby limit progressive infarction. This is generally done surgically. Standard pharmaceutical treatment, such as antiarrhythmics and antithrombotics don't get at the cause of the stroke or the damage caused, but are mostly done to insure adequate cardiac functioning.

**Immunodeficiency**

Immunodeficiency (or immune deficiency) is a condition resulting from a defective immunological mechanism; may be primary (due to a defect in the immune mechanism per se) or secondary (dependent upon another disease process), specific (due to defect in either the B-lymphocyte or T-lymphocyte system, or both) or nonspecific (due to defect in one or another component of the nonspecific immune mechanism). The treatment of "immunodeficiency" generally would be an unprecedented feat. For a compound or genus to be effective against "immunodeficiency" generally is contrary to medical science. The "immunodeficiency" are processes which can take place in virtually any part of the body. There is a vast range of forms that it can take, causes for the problem, and biochemical pathways that mediate the inflammatory reaction. There are dozens of such diseases, which have fundamentally different mechanisms and different underlying causes.

Five classes of primary immunodeficiency diseases have been identified:

1. T-lymphocyte disorders (such as the DiGeorge anomaly and chronic mucocutaneous candidiasis);
2. B-lymphocyte disorders (such as X-linked agammaglobulinemia, common variable immunodeficiency, and selective immunoglobulin A deficiency);
3. Combined T- and B-lymphocyte disorders (such as severe combined immunodeficiency, i.e. SCID, the Wiskott-Aldrich syndrome and ataxia telangiectasia);

4. Phagocytic disorders (such as chronic granulomatous disease) and
5. Complement disorders (such as C2 deficiency and C3 deficiency).

Although the exact etiology of many immunodeficiency diseases is unknown, several etiologic factors have been identified in specific disorders. When normal maturation of the immune system is impaired as in an enzyme or hormone deficiency, immunodeficiency can result. Many immunodeficiency diseases are genetically determined. In some forms of agammaglobulinemia and SCID, an X-linked recessive pattern of inheritance has been demonstrated. In other immunodeficiency diseases, an autosomal recessive pattern of inheritance is evident.

Some immune deficiencies result from environmental factors or occur secondary to other causes. One example is the Acquired Immune Deficiency Syndrome, also known as AIDS, which is caused by the HIV virus. Other immune deficiency diseases occur or are acquired as the result of having cancer, severe nutritional disorders, burns, infections, exposure to radiation or organ transplantation.

### **Syndrome X**

It has been recited a method of treating Syndrome X, but the specification is not enabled for such a scope. Syndrome X is a cluster of risk factors that together, put someone at higher risk of coronary artery disease. These risk factors include: central obesity (excessive fat tissue in the abdominal region), glucose intolerance, high triglycerides and low HDL cholesterol, and high blood pressure.

(B). Scope of Compounds - The scope of the compounds is broad. It is apparent that hundreds of millions of combinations of compounds can be created from the definitions, owing especially to broad scope of A1, A2, and Ar.

(2). Direction of Guidance: The amount of direction or guidance is minimal. The dosage range is 300 fold and hence largely useless. The dosage is completely generic, it is the same regardless of which disorder is being treated.

(3). State of Prior Art: There is no evidence of record that compounds structurally similar to these pyridazine derivative compounds are in use for the treatment of metabolic disorder.

(4). Working Examples: There is no any working example that indicates the inhibition of GSK-3 $\beta$ , which in return is presumed to treat neurodegenerative diseases, strokes, metabolic diseases, syndrome X, immunodeficiency or cancer. There is no data for any actual treatment of disease or of any animal model for treatment of disease.

(5). Nature of the Invention and Predictability: It is well established that "the scope of enablement varies inversely with the degree of unpredictability of the factors involved," and physiological activity is generally considered to be an unpredictable factor. See *In re Fisher*, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970).

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(6). The Relative Skill of Those in the Art: The skill level in this art is too low, because no compound effective against neurodegenerative diseases, strokes, metabolic diseases, syndrome X, immunodeficiency or cancer has ever been found.

In terms of the individual metabolic disorders, this is completely varied. It ranges from areas where the skill level is high, as in carbohydrate metabolic disorders, to a deficiency of the pyruvate dehydrogenase complex, where the skill level is so low that there is no effective pharmacological treatment.

In regard to stroke, the skill level in this is so low. Effective acute drug treatment of the stroke itself has so far proved to be beyond the reach of medical science. Major efforts have certainly been pressed in the area of neuroprotective therapeutics. Those studied have included use of Ca antagonists such as Levemopamil and flunarizine, to suppress neuronal calcium influx; NMDA antagonists (both competitive, such as APV and CPP, and non-competitive such as chlorpromazine, ifenprodil and Mg salts) as well as AMPA and kainate antagonists to block post-ischemic receptor-operated calcium channels; attempts to block arachidonic acid cascade or elimination of its metabolic products with agents such as lipogenase inhibitors and thromboxane; use of free oxygen radical scavengers such as superoxide dismutase, alpha-tocopherol, or allopurinol to inhibit the lipid peroxidation that damages cell membranes, which may indirectly help prevent intracellular calcium overload; anti-edema agents such as corticosteroids; use of 5-HT<sub>1A</sub> receptor agonists to suppress 5-HT concentrations in the hippocampal extracellular

space; use of CRF receptor antagonists to inhibit excitotoxic brain damage; use of serotonin 1A agonists such as ipsapirone, or adenosine modulators such as vinpocetine, to stimulate adenosine, which may act as a protective agent by hyperpolarizing the postsynaptic neuron; use of platelet aggregation inhibitors such as prostacycline and ticlopidine, and other approaches as well. Despite this vast outpouring of research, the skill level in this art is sufficiently low relative to the difficulty of the task that obtaining a neuroprotective treatment of stroke was, as of the filing date, not yet possible. Hence, accomplishing such a goal involves more than routine experimentation. As evidence for this, there is cited Chalmers (TiPS Vol 17, pages 166-172 April 1996), which states flatly on page 170 that, "At present, there are no effective neuroprotective agents that can clinically ameliorate the effects of stroke in humans." For example, Pentoxifylline has been one of the most intensely studied, with dozens of studies published on its properties. It appears to have a wide variety of effects on leucocytes, erythrocytes, neutrophils, plasma fibrinogen levels. These result in a wide-ranging ability to increase blood flow, resulting in effectiveness in some vascular disorders, especially intermittent claudication. Research with different administration methods, or different subcategories of stroke may well result in the discovery of how to get this drug to work, but the slowness and difficulty of this research shows clearly that this involves undue, not routine experimentation. Applicants' compounds have been subjected to far less study.



The relative skill in the treatment of cancer is so low. There never has been a compound capable of treating cancer generally. There are compounds that treat a range of cancers, but no one has ever been able to figure out how to get a compound to be effective against cancer generally, or even a majority of cancers. Thus, the existence of such a “silver bullet” is contrary to our present understanding in oncology. Even the most broadly effective antitumor agents are only effective against a small fraction of the vast number of different cancers known. This is true in part because cancers arise from a wide variety of sources, such as viruses (e.g. EBV, HHV-8, and HTLV-1), exposure to chemicals such as tobacco tars, genetic disorders, ionizing radiation, and a wide variety of failures of the body’s cell growth regulatory mechanisms. Different types of cancers affect different organs and have different methods of growth and harm to the body, and different vulnerabilities. Thus, it is beyond the skill of oncologists today to get an agent to be effective against cancers generally, evidence that the level of skill in this art is low relative to the difficulty of such a task.

(7). The Quantity of Experimentation Necessary: Immense, especially in view of points (1) and (6).

MPEP 2164.01(a) states, “A conclusion of lack of enablement means that, based on the evidence regarding each of the above factors, the specification, at the time the application was filed, would not have taught one skilled in the art how to make and/or use the full scope of the claimed invention without undue experimentation. *In re Wright*,

999 F.2d 1557,1562, 27 USPQ2d 1510, 1513 (Fed. Cir. 1993).” That conclusion is clearly justified here.

### ***Claim Rejections - 35 USC § 112***

6. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-7 and 9-28 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. Claim 1 and claims dependent thereon are rejected because in the definition of R and Ar the phrases “aryl, heterocyclyl and heteroaryl may in turn be at least monosubstituted with C<sub>1</sub>-C<sub>6</sub>alkyl....or OH” or “aryl and heteroaryl may in turn be at least monosubstituted with C<sub>1</sub>-C<sub>6</sub>alkyl....or OH” are not clear. What does “may in turn be” mean? This is not a standard claim language. Is this a proviso that requires that the aryl is monosubstituted by substituents such as alkyl, alkoxy? Then, the claim should be amended so that it reads as proviso.

Note that in the definition of R and Ar, it appears that “at least monosubstituted is referring to C<sub>1</sub>-C<sub>10</sub> alkyl” and not to other definitions of R or Ar. Is this the case?

### ***Objection***


7. Claim 8 is objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including all of the limitations of the base claim and any intervening claims.

***Conclusion***

8. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Kahsay Habte, Ph. D. whose telephone number is (571) 272-0667. The examiner can normally be reached on M-F (9.00AM- 5:30PM).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, James Wilson can be reached at (571) 272-0661. The fax phone number for the organization where this application or proceeding is assigned is (571)-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

  
Kahsay Habte, Ph. D.  
Patent Examiner  
Art Unit 1624

KH  
October 20, 2005